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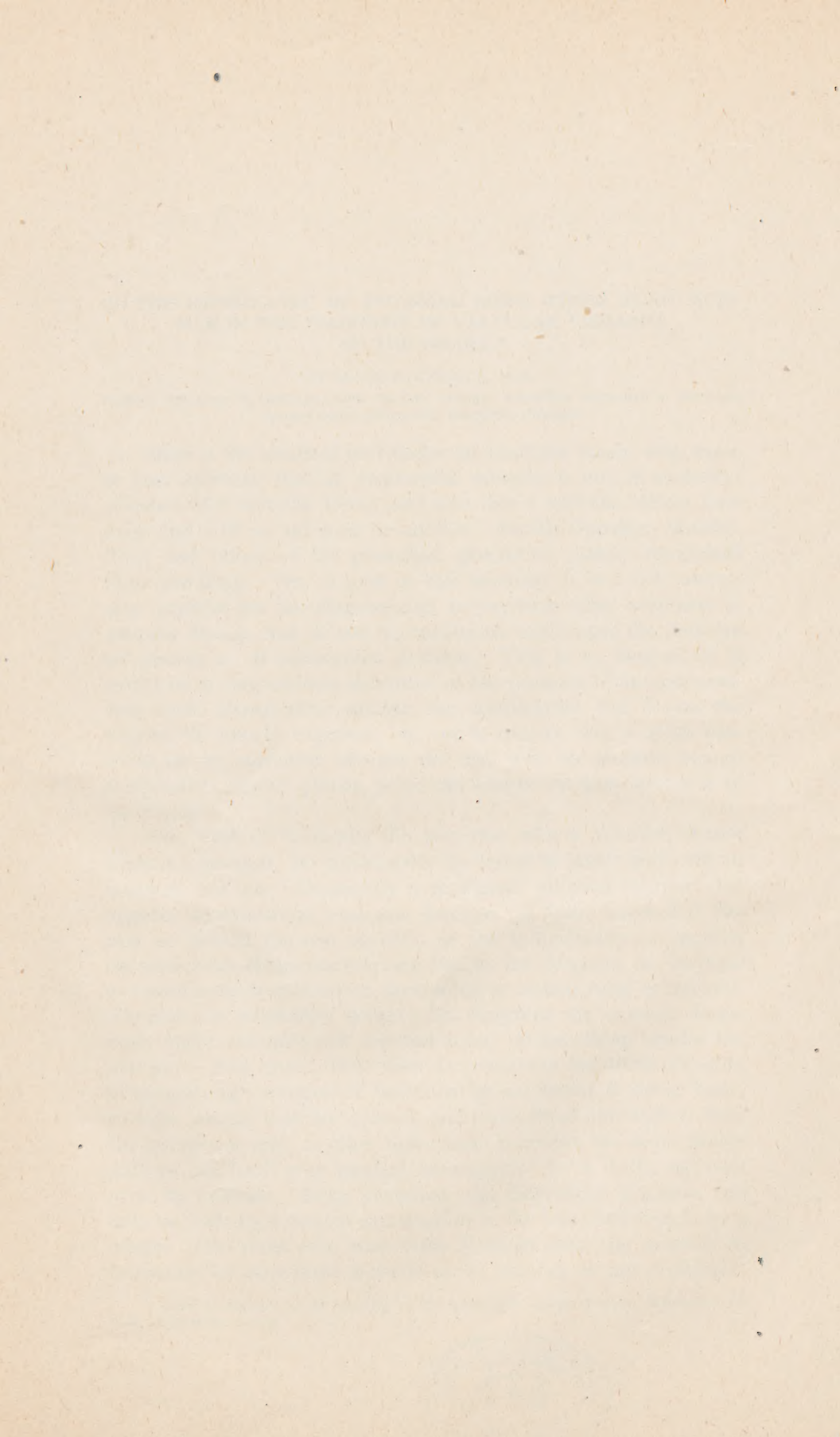
BY

JAMES B. HERRICK, M.D.



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ON THE IMPORTANCE OF PHYSICAL SIGNS OTHER THAN MUR-MUR IN THE DIAGNOSIS OF VALVULAR DISEASES OF THE HEART.¹

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Most of the standard text-books on medicine teach, with more or less clearness, that an endocardial murmur is not, of necessity, evidence of a valvular lesion, and also that a valvular defect may exist and still no murmur be audible. Skoda, Oppolzer, Walshe, Flint and others of the preceding generation plainly recognized these two facts. Yet, in spite of this teaching, it is a not uncommon practice for the diagnostician to overlook other evidences of valvular disease, and to base his conclusion solely upon the presence or absence of an endocardial murmur. This is as illogical as it would be to diagnose scarlatina on the presence of an erythematous rash, disregarding entirely the temperature, the throat, the tongue, the glands, exposure, etc.; or to declare that a given case could not be scarlatina because the rash was not present, though temperature, throat, glands, pulse and tongue all gave evidence of the disease.

For, while it is usually the case that with a valvular disease there is a murmur, yet occasionally the lesion is latent and without murmur, and not infrequently a previously existing murmur disappears as myocardial weakness develops (as from myocardial disease or toward the end of life), or the individuality or peculiar characteristics of the murmur are lost to the listening ear in rapid or tumultuous heart's action, pericardial or pleural rubs, or tracheal, bronchial, or pulmonary noises. To recognize the valvular lesion under these circumstances, one has to rely on something besides the murmur. And again, while there is commonly but little difficulty in recognizing a murmur as functional or accidental, it being basic, systolic, neither loud nor musical, and transmitted but slightly from the pulmonary area, at other times these murmurs are more widely diffused, are loud, even musical, accompanied by a thrill, and may even be diastolic. Here, therefore, the differential diagnosis can only be made by a careful examination of the heart and vessels as a whole. One must rely upon other findings than the murmur to determine the important question as to whether or not a valvular

¹ Read in abstract at the meeting of the Mississippi Valley Medical Association, St. Paul, September 16, 1896.



lesion is present. In some doubtful cases the therapeutic test of rest, iron, digitalis, will be of great aid in clearing up a diagnosis.

It is the object of this paper to emphasize the value of paying attention to signs other than the murmur, and to show that, while of great aid in diagnosis, the endocardial murmur can only be estimated at its true worth when considered in the light of the other physical findings.

It is important in this connection to keep clearly in mind certain pathological consequences of valvular lesion. For it is as much by the recognition of these changes in the anatomical or physiological conditions of the circulatory apparatus, as by the murmur, that the presence or absence of a valvular disease can be predicated.

1. Every valvular lesion, whether of obstruction or insufficiency, must result in hypertrophy and dilatation of the heart behind (as regards the blood-current) the valve diseased. Hypertrophy of some portion of the heart, therefore, may be one of the strongest evidences of valvular disease in front of the hypertrophy. Absence of hypertrophy may prove a murmur functional or accidental.

2. An increase in the tension of the pulmonary circulation follows any valvular lesion (in reality an obstruction) at the mitral orifice. Later, also, any aortic disease will produce the same result. Increased pulmonary tension shows in increased force and intensity of the pulmonic second tone, a point first emphasized by Skoda. Yet, later, a failing right ventricle may cause lowering of pulmonic pressure and therefore weak pulmonic second tone. Nowhere is the value of examination of the pulmonic valvular sound more clearly shown than in mitral stenosis, where, not infrequently, the murmur is absent. Here the snappy, intense pulmonic second shows in striking contrast to the aortic second that is weak because of low systemic arterial tension, and this may be the convincing diagnostic sign of mitral obstruction.

3. Stenosis of the orifices of the left heart means smaller amount of blood in the general arterial circulation, and therefore lessened arterial tension as shown by the pulse. The small pulse of aortic or mitral stenosis illustrates this point.

4. Failure of the right heart is followed by general venous congestion with varying phenomena, *e.g.* venous pulse, hepatic and general portal congestion, anasarca of the lower portion of the body.

If the examiner keeps these facts clearly in mind, he will find much more than the murmur to seek for. He will, in fact, defer auscultation until the last, and will aim by inspection, palpation and

percussion to learn what he can of the size of the heart and its method of work.

Inspection.—Hypertrophy of the left heart is commonly recognized by the heaving, forcible apex-impulse. The massive hypertrophy of aortic disease, with heaving apex far to the left and downward, may lead one to suspect the valve diseased, even on inspection, and, by noting the peripheral pulse and the frequency of beat, a regurgitant or obstructive lesion may be surmised. The closure of the pulmonary semilunars occasionally causes a perceptible impulse when the overlying lung is retracted or pushed aside by an enlarged heart and when the force of the semilunar closure is exaggerated. An enlarged right heart may also produce a diffused impulse between the sternum and left nipple, and epigastric pulsation is rightly regarded as of great value as an indication of right-heart dilatation. A failing right heart with incompetent tricuspid means not alone epigastric pulsation, but a true ventricular, systolic, positive, jugular pulse. And not infrequently hepatic pulse, to be distinguished from the communicated hepatic heaving, is an accompaniment of tricuspid insufficiency. Even the peripheral veins may, from a similar cause, show pulsation.

The behavior of the arteries in valvular lesions is of great diagnostic value, particularly in aortic regurgitation. In fact, it is no great feat to make a diagnosis of aortic regurgitation by inspection alone, or by inspection and palpation. The suddenly overfilled, bounding, tortuous arteries, which as suddenly collapse, taken with the signs of enlarged left heart, and perhaps with a capillary pulse, can scarcely mean anything else. Most liable to confuse would be the hypertrophied heart and tortuous vessels of arteriosclerosis and contracted kidney. The feel of the pulse will generally enable the differential diagnosis to be made.

The bulging præcordia of children with hypertrophy should be noticed. And alterations of the chest-wall may call attention to retracted lungs or to emphysema, that may explain an accidental murmur, or dilatation with relatively insufficient valves. The existence of anemia, cyanosis, edema, will not escape attention.

Palpation.—Palpation seeks mainly to confirm the results obtained by inspection. The location, strength and diffusion of the apex-impulse, epigastric pulsation, and hepatic pulse, are thus readily recognized. The diastolic shock over the pulmonary valves may furnish conclusive evidence of pulmonary congestion in consequence of left-heart valvular disease. A presystolic apical thrill is generally indicative of mitral stenosis. Systolic thrills, while usu-

ally due to organic disease, are also found associated with functional murmurs.

The value of careful palpation of the pulse need not be dwelt upon at any length. While it is asserting too much to say that each valvular lesion has its own peculiar pulse, one may safely assert that not infrequently the character of the pulse lends strong confirmatory evidence of a particular valvular lesion. The Corrigan pulse of aortic insufficiency is a good illustration. A slow pulse that is small and weak in spite of the fact that there is greatly hypertrophied and laboring left ventricle, speaks for aortic stenosis. A small, irregular, usually rapid pulse is commonly found in mitral stenosis. The pulse of mitral regurgitation is not so often of diagnostic value, as it frequently differs very little from the normal.

Percussion.—Without proof of cardiac enlargement, suspicion is thrown at once upon the organic valvular origin of an endocardial murmur. It is necessary, too, to determine what parts of the heart are enlarged, whether right or left, the auricle or the ventricle. Thus a diastolic left-heart murmur without left ventricular hypertrophy, but with right-heart enlargement, speaks against aortic regurgitation and for mitral stenosis. Again, percussion, with the other methods of physical diagnosis, enables one to exclude extra-cardiac causes for murmurs such as might arise in a heart dislocated by retracting extra-cardiac tissue or by pressure (*e.g.*, aneurism, tumor, fluid, air, or retracted lung). A slur is sometimes cast upon cardiac percussion because two examiners do not agree absolutely in their markings of the cardiac outlines obtained by percussion. This is because the deep cardiac dullness is relative, and each ear has an individual standard of relative dullness. But great practical uniformity in results will be found on comparing the findings of two skilled examiners—that is to say, both will agree that there is left ventricular hypertrophy, right-heart dilatation, etc.

Auscultation.—On auscultation more is to be listened for than the endocardial murmur. The value of an accentuated pulmonic second tone as an evidence of pulmonary engorgement, such as comes with left-heart valvular disease, has been referred to, but will bear repetition. An accentuated aortic sound may call attention to the kidney as the cause of the increased systemic tension. A weak aortic sound may be an indication of insufficient supply of blood to the aorta because of mitral or aortic obstruction. Reduplication of basic second tones may point to valvular disease, as in mitral stenosis. A sharply accentuated, snappy first sound at the apex is common in mitral stenosis. The tones in the cervical and peripheral

vessels are not without value in diagnosis—*e. g.*, the well known peripheral tones in aortic regurgitation.

Illustrations of the truth of the foregoing statements could be multiplied from the writings of others, or from one's own practice. I have several times exhibited to classes cases of aortic regurgitation, and have been able to bring so clearly to the attention of the students the cardiac hypertrophy, the accentuated pulmonic sound, the throbbing tortuous arteries with water-hammer pulse, the Quincke pulse, the peripheral tones, that the students have made the probable diagnosis without one word said about murmur. In the same manner I have had them make the diagnosis of mitral stenosis by accentuated and palpable pulmonic second, the right-heart enlargement, the abrupt first sound, and the rapid, small, irregular pulse. That the murmur in mitral obstruction is very frequently absent, is the testimony of nearly all writers. This is so not alone during terminal asthenia, but when for any cause the left auricle becomes weak. In two Cook County Hospital cases that I recall there was a common agreement among several physicians who examined them that mitral stenosis existed, though at times the presystolic or diastolic murmur with the thrill could not be found. I have a patient in private practice in whom I have never found a distinct presystolic murmur; but there is little doubt that there is mitral constriction, because of the other signs just enumerated.

Tachycardia, pulmonary or bronchial sounds, or some other predominant murmur, may drown any feeble murmur. Under these circumstances one has to rely solely upon the accompanying evidences of valvular disease. A young woman six months pregnant, who knew she had heart disease, came to the County Hospital giving a history of rather suddenly developing dyspnea with slight hemoptysis. She was moderately cyanotic, orthopneic, with numerous râles of edema, and a small dull area with diminished vesicular sounds over the lower right lung posteriorly, presumably an infarct. The heart was arrhythmic, beating tumultuously at about 140 to 150. I was unable to analyze the murmurs heard; but a probable diagnosis of double mitral lesion was made, not so much from the enlarged right heart and accentuated and palpable pulmonic second as from the abrupt, snappy and loud first tone at the apex. When under morphine, rest and digitalis the heart had quieted down, there were made out typical systolic and diastolic apical murmurs. These cases merely serve as illustrations of the fact that we often can, and sometimes must, make a diagnosis of valvular lesion without regard to murmur.

By failing to give proper regard to the secondary signs of valvular disease, one can easily err in calling a functional murmur an organic one. The danger is greatest, I believe, in the murmurs arising during acute infectious diseases, such as rheumatism, typhoid, pneumonia, chorea. I recall two such errors, corrected by autopsy: one in typhoid and one in tuberculosis. It is not always possible to differentiate between the inorganic murmur and that of an acute endocarditis; yet time and a due regard to the secondary signs of valvular disease will usually clear up the diagnosis.

Nearly forty years ago Walshe clearly recognized the fact of the temporary disappearance of organic murmurs when he said: "Every necessary organic condition of a heart murmur may be present, and yet the resulting murmur be soft, if the current be feeble—nay, the murmur may be wholly deficient." And again: "Excite a tranquil heart, and a murmur previously almost inaudible becomes distinct; weaken the energy of the cardiac contraction by digitalis [!] or aconite, and the converse result follows."¹

All of the older writers on diseases of the heart recognized—some more clearly than others—the value of secondary signs in determining whether or not a murmur was the so-called anemic, hemic, or functional murmur. So these points are not new. But they are too often forgotten in practice. That the profession is becoming alive to their importance is shown by the frequent reference to these topics in current literature, and incidentally by the fact that at this meeting there are presented three papers upon practically this same subject.

In this paper the intention has not been to undervalue the importance of the endocardial murmur, but to insist that it is only by the complexus of signs and symptoms that an accurate diagnosis is made in these cases. With combined and complicated murmurs, and with tumultuous, rapid and very weak heart, one has often to hesitate as to the nature of the lesion or to confess, with Fraentzel, inability to solve the problem as to the exact character of the heart lesion. But the more one learns about the heart, the more he realizes the truth there is in Fraentzel's rather extravagant statement, that of all the evidences of heart disease the least valuable is the endocardial murmur.²

¹ Walshe: *On Diseases of the Heart*, pp. 80 and 83. Philadelphia, 1862.

² Fraentzel: *Die Krankheiten des Herzens*, ii, p. 84. Berlin, 1891.

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"Medicine"

THE appearance of the first number of the second volume of *Medicine*, the monthly medical journal edited by Harold N. Mayer, M. D., of Chicago, and published by George S. Davis of Detroit, prompts the just comment that no medical journal that we have seen at all approaches it in newness and beauty of typography or, in general, in the quality of the printer's and publisher's work. In contrast with the host of so-called medical journals, printed poorly upon inferior paper, it is a keen pleasure to pick up a number of *Medicine* and realize what an important part the mere mechanical work put upon a journal plays.

It must not be concluded, however, that charm of appearance is the only good quality of *Medicine*. It is well edited and its pages are clean and free from any objectionable feature. It deserves a place among the very foremost of American medical journals and we sincerely wish it a full measure of success. The general quality of medical journals of this country is so low that the success of all the better ones is to be devoutly wished for by all physicians who feel a pride in American medicine.

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